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NARRATIVE REVIEW

Bedside thoracic echography in ICU: new insights for acute circulatory and respiratory failure

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Abstract

In intensive care, hemodynamic and respiratory failure are the two main types of organ failure and are frequently associated. In cases of shock and/or acute dyspnea, thoracic ultrasound involves the simultaneous use of lung ultrasound and transthoracic echocardiography. While rigorous clinical assessment remains fundamental, the current literature shows that adding thoracic ultrasound to conventional examination improves diagnostic accuracy and enables rapid, adequate therapeutic decisions during shock and/or acute respiratory failure in a non-invasive way. However, its impact on survival is still being debated. This narrative review summarizes the criteria that help identify the five main hemodynamic profiles during shock, as well as the endpoints that allow discrimination between cardiogenic pulmonary edema, acute respiratory distress syndrome, pneumonia, pleural effusion and pneumothorax. It also proposes pragmatic algorithms for managing shock and/or respiratory failure at the bedside. Current data suggest that artificial intelligence and automated measurements will greatly improve the diagnostic performance, reproducibility, ease of use, and teachability of thoracic ultrasound in the near future.

Keywords: Lung ultrasound, Echocardiography, Point-of-care ultrasound, Shock, Acute circulatory failure, Acute respiratory failure, Echography, Ultrasound

Introduction

Thoracic ultrasound combines transthoracic echocardiography (TTE) and lung ultrasound (LUS). In the intensive care unit (ICU), it enhances diagnostic accuracy for all shock types and acute dyspnea, especially in differentiating cardiogenic causes from respiratory diseases [1–3]. Thoracic ultrasound is non-invasive, widely available, and shortens time to diagnosis, treatment, and ICU stay [2, 4]. Guidelines recommend early thoracic ultrasound use to guide hemodynamic management, including fluid and vasopressor therapy [5–7]. Although its impact on mortality is debated, thoracic ultrasound correlates with prognosis in ICU patients [2, 8]. This narrative review outlines key thoracic ultrasound criteria for bedside diagnosis and management of shock and dyspnea.

The main echo techniques, abbreviations, and normal values are detailed in supplementary material S1. The following article discusses the more frequent situations. In case of severe congenital disease, severe valve dysfunction, prosthetic valve, structural cardiac disease (severe hypertrophic obstructive cardiomyopathy, restrictive cardiomyopathy, cardiac transplant recipients, LV assist device), the proposed algorithms cannot be applied. In this case, however, lung ultrasound remains valid. It is

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also important to note that most of the published literature is based on observational studies, or non-blinded controlled studies which, therefore, carry methodological limitations.

Methodology

This narrative review was conducted by a multidisciplinary panel of international experts, all of whom have published extensively in peer-reviewed journals (Q1/Q2 quartiles) and represent diverse countries and geographic regions. The aim was to provide a didactic overview of thoracic point-of-care ultrasound (POCUS) in the context of acute circulatory and respiratory failure.

Literature search strategy

A systematic search was performed using PubMed and Cochrane databases to identify relevant studies published between January 2000 and 2025. The search was restricted to full-text articles written in English. The following MeSH terms (PubMed) and free-text keywords were combined to maximize sensitivity and specificity:

- Core terms: *"ultrasound," "diagnostic imaging," "point-of-care ultrasound," "chest," "thorax," "circulatory collapse," "respiratory failure," "acute care," "emergency medicine," "critical care."*
- Chapter-specific terms: for example, *"lung ultrasound"* AND *"pulmonary edema," "focused cardiac ultrasound"* AND *"shock,"* etc.

Take home message

Thoracic ultrasound is the most performed point-of-care ultrasound examination in ICUs. Recent studies suggest that using it may improve patient prognosis. This paper aims to summarize recent developments and future directions in this area, with a particular focus on the potential contribution of artificial intelligence. It also provides practical algorithms for use at the bedside in cases of acute respiratory or circulatory failure.

Thoracic ultrasound to identify the cause of shock and to guide resuscitation (Fig. 1, Table 1)

Profile 1: severe hypovolemia, fluid responsiveness assessment Clinical assessment alone is unreliable for predicting fluid responsiveness, only ~ 50% of fluid challenges proving effective [13]. Thoracic ultrasound offers a more accurate assessment, identifying both fluid responsiveness and risks of fluid overload.

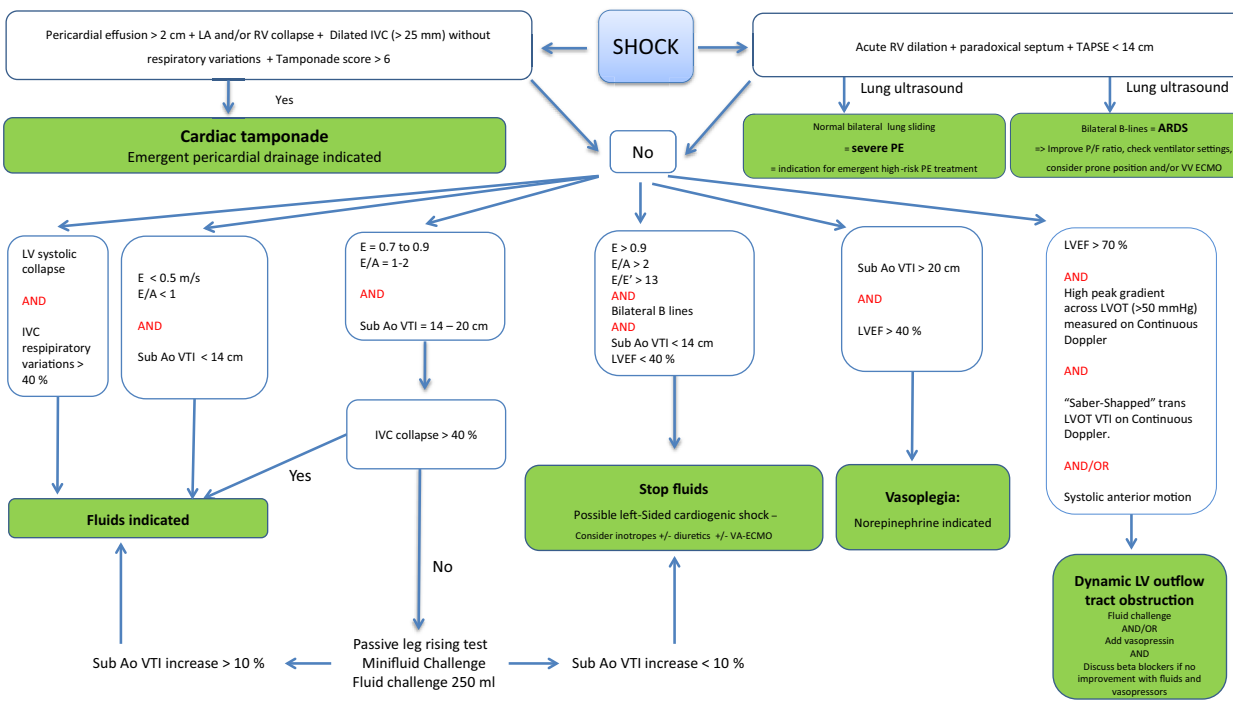

Fig. 1 Thoracic ultrasound for shock management. E = maximal velocity of mitral E-wave recorded on pulse-wave Doppler. *A* maximal velocity of mitral A-wave recorded on pulse-wave Doppler, *E'* maximal velocity of mitral E'-wave recorded on tissue Doppler at the mitral annulus, *Sub Ao VTI* sub-aortic velocity–time integral, *LVEF* left ventricle ejection fraction, *IVC* inferior vena cava. Doppler. *RV* right ventricle, *RA* right atria, *LVOT* left ventricle outflow tract, *PLR* passive leg rising, *TAPSE* tricuspid annulus plane systolic excursion, *PE* pulmonary embolism

In severe hypovolemia, the combination of systolic left ventricular (LV) collapse ("kissing LV") (Video S1) and > 40% respiratory variation in inferior vena cava (IVC) (Video S2) diameter suggests the need for urgent fluid resuscitation or transfusion [14].

The IVC respiratory variations alone are unreliable for predicting fluid responsiveness in both ventilated and non-ventilated [15]. In mechanically ventilated patients, sensitivity and specificity are, respectively, 0.67 and 0.68 (area under the ROC curve = 0.79). In non-ventilated patients, sensitivity and specificity

Table 1 Critical thoracic ultrasound values for circulatory shock management

Clinical syndrome	Echocardiography	Lung ultrasound	Diagnosis
Profile 1: fluid responsiveness assessment			
Profile 1a	LV systolic collapse and IVC/SVC respiratory variations > 40%	Normal or abnormal	Severe hypovolemia Fluid loading indicated
Profile 1b	E < 0.7 m/s and SubAo VTI < 14 cm		Probable hypovolemia, fluid loading indicated
Profile 1c	0.1 < E < 1 m/s and 0.5 < E/A < 2 and 14 < Sub Ao VTI < 20 cm		Gray zone: fluid responsiveness assessment with either: 250 ml fluid infusion over 10 min or PLR test or mini-fluid challenge
Profile 2: left-sided cardiogenic shock			
Profile 2a	LVEF < 40% and Sub Ao VTI < 14 cm and E/E' ratio > 14	Normal	Left-sided cardiogenic shock without pulmonary edema. Inotropes indicated
Profile 2B	LVEF < 40% and Sub Ao VTI < 14 cm and /E' ratio > 14	Bilateral B-lines No spared areas Non-irregular pleura	Left-sided cardiogenic shock with pulmonary edema Inotropes and diuretics indicated
Profile 3: acute right ventricle failure			
Profile 3a	Dilated RV and paradoxical septum and 2.5 < TR V_{max} < 3.5 m/s and TAPSE < 14 cm or reduced RVEF (< 40%)	Normal	High-risk pulmonary embolism Consider fibrinolysis[9] Or Catheter-directed thrombectomy [10] Or Surgical thrombectomy [9] Consider VA-ECMO [11] if no hemodynamic improvement with inotropes and vasopressors
Profile 3b	Dilated RV and paradoxical septum and 2.5 < TR V_{max} < 3.5 m/s and TAPSE < 14 cm or reduced RVEF (< 40%)	Bilateral B-lines and spared areas and irregular pleura and/or consolidations	ARDS with severe hypoxic hypoxemic pulmonary vasoconstriction Consider all means to increase P/F ratio: prone position, inhaled nitric oxide, and/ or VV ECMO
Profile 4: cardiac tamponade	Pericardial effusion > 2 cm and RV and/or RA collapse and dilated IVC > 25 mm without respiratory variation	Normal or abnormal	Emergent pericardial drainage indicated If doubt, calculate tamponade score [45] and perform emergent pericardial drainage if score > 6
Profile 5: left ventricular hyperkinesia with LVOT obstruction	LVEF > 70% and high peak gradient across LVOT (> 50 mmHg) measured on continuous Doppler and "Saber-Shaped" trans LVOT VTI on continuous Doppler	Normal or abnormal	Norepinephrine and fluid loading Add vasopressin if no improvement Consider adding infusion of short-acting beta blocker if no improvement with norepinephrine, fluids and vasopressin

E = maximal velocity of mitral E-wave recorded on pulse-wave Doppler. A = maximal velocity of mitral A-wave recorded on pulse-wave Doppler. E' = maximal velocity of mitral E'-wave recorded on tissue Doppler at the mitral annulus. SubAo VTI = sub-aortic VTI. LVEF: left ventricle ejection fraction. IVC = inferior vena cava. SVC = superior vena cava. TR = tricuspid regurgitation recorded on continuous Doppler. RV: right ventricle. RA = right atria. LVOT = left ventricle outflow tract. PLR = passive leg rising. RVEF = right ventricle ejection fraction. TAPSE = tricuspid annulus plane systolic excursion

are, respectively, 0.52 and 0.77 (area under the ROC curve = 0.76) [15]. In spontaneous ventilation, a high value (> 40%) of IVC collapsibility index (maximal diameter–minimal diameter/maximal diameter) is predictive of fluid responsiveness. A value < 40% cannot exclude preload dependency [16, 17]. In ventilated patients, IVC respiratory variations depend on tidal volume, which is the main determinant of cardiorespiratory interactions [18]. In case of low tidal volumes (< 6 ml/kg), the area under the ROC curve for predicting fluid responsiveness is poor (0.70) [18]. In patients with tidal volume > 8 ml/kg, the area under the ROC curve is 0.88 (sensitivity 0.80, specificity 0.94) [18]. A transient increase in tidal volume from 6 to 8 ml/kg can help to detect fluid responders.

When these signs are absent, a sub-aortic velocity time integral (VTI) < 16 cm associated with a mitral E-wave velocity < 0.7 m/s (indicating both low stroke volume and low LV filling pressure) (Fig. S1, Tables S1 and S2) can help predict fluid responsiveness [19].

Some ultrasound criteria can be used to avoid or stop fluid administration.

1. A sub-aortic VTI < 14 cm combined with LVEF < 40% (or surrogate as low S' wave velocity (< 6 cm/s) or low mitral annulus plane systolic excursion (MAPSE) (Fig. S2) < 7 mm suggests severe LV failure and a high risk of pulmonary edema, especially if lung ultrasound shows bilateral B-lines [19] (Fig. S3, Video S3).
2. Two or more of the following indicate high LV filling pressure: dilated left atrium (> 32 ml/m^2), E/E' ratio > 14, tricuspid regurgitation velocity > 2.8 m/s, E-wave deceleration time < 140 ms during sinus rhythm; E/E' ratio > 11, tricuspid regurgitation > 2.8 m/s; E-wave deceleration time < 160 ms, mean E-wave velocity > 1 m/s, during atrial fibrillation [20–22].

The most reliable way to assess fluid responsiveness is by measuring changes in sub-aortic VTI.

- Conventional fluid challenge: 4 ml/kg over < 15 min; a ≥ 10–15% VTI increase defines fluid responsiveness [23].
- Mini-fluid challenge: 100 ml in 1 min; a ≥ 10% VTI increase is predictive, especially in patients at risk of fluid overload (e.g., ARDS, septic shock) [24]
- Passive leg raising (PLR): simulates a 300 ml fluid bolus by elevating legs to 45° from semi-recumbent. A ≥ 10–15% VTI increase within 1 min confirms responsiveness. This maneuver is applicable in both ventilated and non-ventilated patients, including pregnancy [25, 26].

In stable patients, even responders, fluid administration must be avoided to limit fluid overload that negatively affects prognosis.

Profile 2: left ventricle cardiogenic shock Left ventricle cardiogenic shock is a life-threatening condition caused by impaired left ventricular function, leading to low cardiac output and organ hypoperfusion [27, 28]. Low LV ejection fraction is classically measured by Simpson's method but surrogate as S′ velocity or MAPSE are often used in ICU because they are easiest and faster to record (Fig. S1). Low cardiac output is assessed by recording sub-aortic VTI, which directly reflects stroke volume (Fig. S1). Cardiogenic shock carries a high mortality (30–40% in the short term, > 50% at 1 year) [28]. Point-of-care echocardiography is crucial for early diagnosis and management. It allows intensivists to rapidly identify severe LV dysfunction and possible underlying causes, such as valve disease, wall motion abnormalities, or cardiomyopathies. If left-sided cardiogenic shock is suspected, a full advanced TTE or transesophageal echocardiography (TEE) should be performed [29]. Doppler measurements help estimate stroke volume, cardiac output, and left atrial pressure [30]. Although it has only been validated in septic shock, a low oxygen delivery (DO_2 < 4 ml/kg/min) may indicate severe left ventricular cardiogenic shock, warranting prompt therapeutic intervention. Echocardiography guides the need to initiate inotrope therapy, monitors response through VTI measurements, and helps determine mechanical circulatory support or cardiac catheterization indications.

Profile 3: acute right ventricular (RV) failure and acute pulmonary embolism TTE is particularly useful for assessing RV function, even at a basic level [31, 32]. However, RV failure remains hard to define at the bedside [33]. Indeed, RV failure even with RV dilatation can occur with normal CVP [34]. Two main approaches exist: (1) identifying RV systolic dysfunction using TAPSE, peak systolic velocity of tricuspid annulus on tissue Doppler or RV fractional area change [35] (Fig. S2); (2) defining RV failure by RV dilation (Fig. S4) plus elevated CVP (> 8 mmHg), indicating systemic congestion [36, 37].

In ARDS and pulmonary embolism (PE), RV dysfunction may appear as acute cor pulmonale, characterized by RV dilatation (RV/LV > 0.6 or > 0.8 according to studies) and paradoxical septal motion (Fig. S4, Videos S4/S5), due to high RV afterload [38]. The increase in the afterload may be due to obstruction either proximal (PE) or distal pulmonary capillaries (ARDS). *Cor pulmonale* is

present in 22% of moderate-to-severe ARDS cases and worsens outcomes [39]. Implications are: (1) in *cor pulmonale* with elevated CVP, avoid fluids, even in shock [37, 40]; (2) high driving pressure (≥ 18 cmH_2O), hypercapnia ($PaCO_2 \geq 48$ mmHg), and severe hypoxemia ($PaO_2/FiO_2 < 150$) increase *cor pulmonale* risk and should be corrected [39]; (3) prone positioning improves both lung mechanics and RV function; (4) severe RV dilation associated with high driving pressure can guide selection for Veno-Venous ExtraCorporeal Membrane Oxygenation (VV ECMO), allowing very low tidal volume and subsequent drastic reduction of driving pressure [41]. Future studies will help evolve toward more precise and individualized allocation of this resource. In critically ill patients, the association of TTE, LUS, and venous ultrasound helps confirms (RV failure and venous thrombosis) or rules out (no RV failure, no venous thrombosis, alternative diagnosis) the diagnosis of PE with an area under the curve at 0.89, and classifies the risk [9, 42, 43]. A mobile clot in the right heart also confirms PE without need for CT. In other clinical scenarios or in the absence of one of these signs, a CT scan is necessary. In high-risk PE with shock and RV dysfunction, urgent reperfusion is recommended. Fluid overload may worsen RV failure, especially in case of severe RV dilation [12].

Profile 4: cardiac tamponade Cardiac tamponade is a clinical syndrome typically associating shock to rising jugular venous pressure and muffled hearts sounds (Beck's triad). A pulsus paradoxus > 10 mmHg is also specific. However, the sensitivity of clinical diagnosis remains low [44]. The use of cardiac tamponade score improves diagnostic performance and helps determine the need for pericardial drainage. [45].

This score [45] is based on anamnestic criteria (malignant disease, tuberculosis, recent radiotherapy, recent viral infection, recurrent pericardial effusion, terminal renal failure, immunodepression, autoimmune or thyroid disease), clinical criteria (hypotension, pulsus paradoxus > 10 mmHg, dyspnea, orthopnea, chest pain, pericardial friction rub), and imaging criteria (cardiomegaly on chest X-ray, electric alternans, microvoltage on ECG and echocardiography signs). A total tamponade score > 6 indicates emergent pericardial drainage [45].

The echocardiography criteria of cardiac tamponade are:

- Circumferential pericardial effusion > 2 cm (score = 3).
- Right atria systolic collapse > 1/3 of cardiac cycle (score = 1).
- IVC diameter > 25 mm with less than 50% of respiratory variations (score = 1.5).
- Right ventricular diastolic collapse (score = 1.5).
- Left atria collapse (score = 2).
- Mitral/tricuspid respiratory variation flow (score = 1).
- Swinging heart (score = 1).

In a recent retrospective study including 239 patients who underwent pericardial drainage within 48 h of admission, the echocardiographic signs were: 69.1% right ventricular diastolic collapse, 62.3% respiratory variation of mitral valve inflow velocities, 56.4% inferior vena cava plethora, and 53.4% right atrial systolic collapse [46].

A simplified score was recently proposed to predict the need for pericardial drainage, based on four variables: systolic blood pressure < 100 mmHg (1.5 points), effusion diameter (1–2 cm = 0 points, 2–3 cm = 1.5 points, > 3 cm = 2 points), right ventricular diastolic collapse (2 points), mitral inflow velocity respiratory variation > 25% (1 point). The need for pericardial drainage within 24 h was 8.1% for a score < 2, 63.8% for a score 2–4, and 93.7% for a score > 4 points. The area under the curve of this score was > 0.90 [47].

Profile 5: left ventricular hyperkinesia with obstruction in ICU patients LV hyperkinesia with dynamic obstruction is a relatively frequent and often underrecognized hemodynamic condition in the ICU, particularly in early septic shock—reported in up to 22% of patients [5, 48]. This condition is associated with increased mortality, especially when the ejection fraction (EF) exceeds 70% [49, 50]. Obstruction can occur at different levels of the LV (apex, mid-ventricle, or LV outflow tract) and typically results from a combination of anatomical predispositions—such as LV hypertrophy, hypertrophic cardiomyopathy, Takotsubo syndrome, or prosthetic valve malposition—and precipitating factors like hypovolemia, vasoplegia, use of inotropes and vasodilators (e.g., dobutamine, nitrates) [48, 51–53].

Pulmonary artery catheter findings may be misleading, mimicking cardiogenic shock with low cardiac output and elevated filling pressures. In contrast, echocardiography remains the most reliable bedside diagnostic tool. Key echocardiographic findings include: hyperdynamic LV function (LVEF > 70%, systolic collapse of left ventricle); systolic anterior motion of the mitral valve (displacement of the distal portion of the anterior leaflet of the mitral valve toward the left ventricular outflow tract obstruction) (Fig. S5a and 5b, Video S6); sub-aortic VTI non-recordable with pulse-wave Doppler and dagger (or saber)-shaped flow on continuous wave Doppler in the left ventricle outflow tract (Fig.S6); mitral regurgitation (Fig. S6); premature aortic valve closure. Color Doppler and pulsed Doppler help localize the obstruction along the LV cavity, while continuous wave Doppler confirms the pressure gradient across the obstruction [48].

Management focuses on treating the underlying pathophysiology: volume resuscitation for hypovolemia; vasopressors (e.g., vasopressin) to restore afterload in case of low peripheral vascular resistance; careful use of short-acting beta-blockers once volemia and vascular tone are restored. Early detection through TTE and tailored hemodynamic treatment using the previously mentioned therapies can restore hemodynamics and may improve prognosis [48, 54–56].

Thoracic ultrasound in acute respiratory failure (Fig. 2)

In normal conditions, lung ultrasound shows lung sliding (horizontal hyperechoic line with lateral movements synchronized with ventilation), associated with A-lines artifacts (repeated horizontal linear hyperechoic lines) (Fig. S7) (Videos S7, linear probe. Video S8, convex probe). Lung ultrasound requires exploring six regions of interest on each side (Fig. S8). The 12 regions of interest are needed to not miss focal anomalies and are required to establish the lung ultrasound score.

How to diagnose cardiogenic pulmonary edema In patients with acute respiratory failure, LUS can accurately confirm or exclude cardiogenic pulmonary edema and assess its severity [57]. The hallmark LUS finding is a diffuse interstitial pattern, characterized by multiple bilateral B-lines [58]. When left-sided heart failure causes pulmonary edema, this pattern should correlate with elevated filling pressures seen on TTE. If diffuse bilateral B-lines are absent, especially in severe respiratory failure, cardiogenic pulmonary edema can be ruled out, with near 100% sensitivity and negative predictive value. In severe cases, B-lines typically appear in anterior–superior zones. In milder or chronic cases, B-lines may only be seen laterally or postero-laterally [59]. In both, the distribution is usually homogeneous and follows gravity—from dependent to non-dependent zones [60].

The typical distribution of the B-lines pattern helps differentiate cardiogenic from non-cardiogenic causes (e.g., ARDS, pulmonary fibrosis), particularly when combined with other LUS signs like a regular pleural line and absence of sub-pleural consolidations [60]. Pleural effusion, if present, is usually anechoic and may cause compressive atelectasis. Complex or echogenic effusions suggest alternative or coexisting conditions.

A combined lung and cardiac ultrasound approach is recommended: LUS defines the extent of pulmonary congestion, while TTE helps identify the underlying cardiac cause (Fig. 2).

Lung ultrasound for the diagnosis of pneumonia LUS, when integrated into clinical practice, improves the accuracy and speed of diagnosing pneumonia in patients with

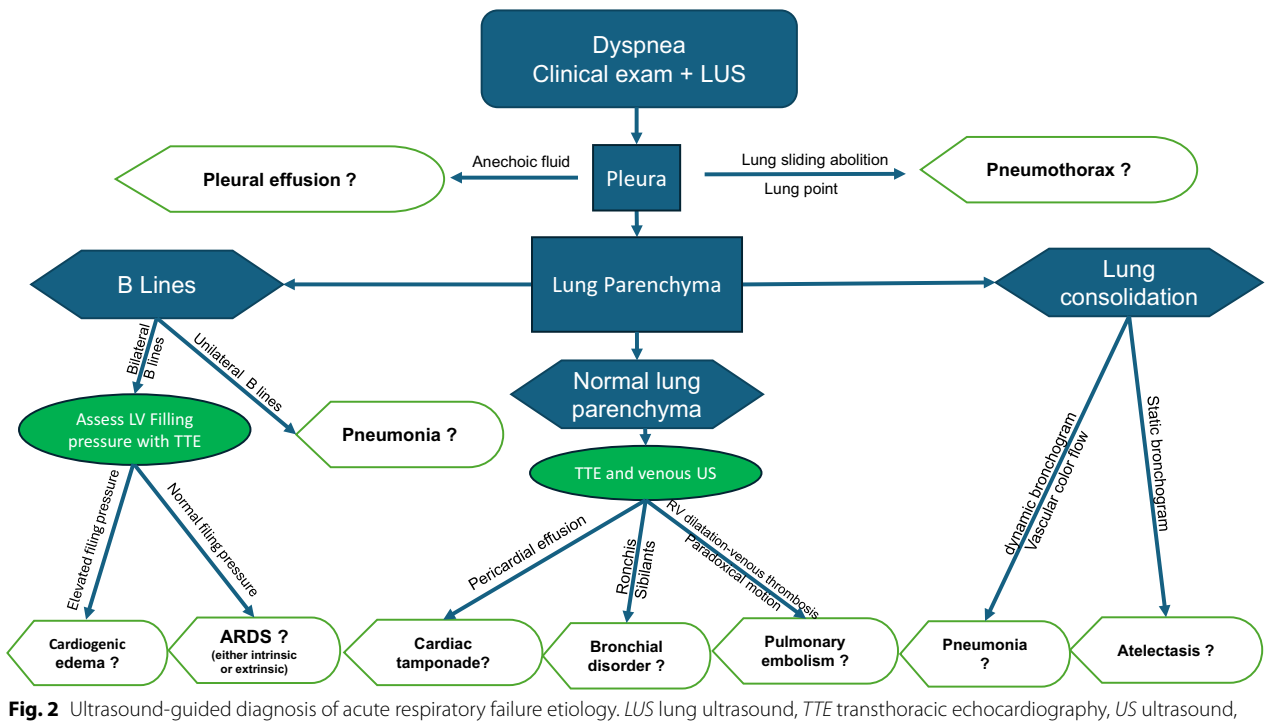

Fig. 2 Ultrasound-guided diagnosis of acute respiratory failure etiology. *LUS* lung ultrasound, *TTE* transthoracic echocardiography, *US* ultrasound, *ARDS* acute respiratory distress syndrome, *LV* left ventricle, *RV* Right Ventricle

acute respiratory failure [60]. In community-acquired bacterial pneumonia, LUS typically shows focal B-lines, irregular pleura, and sub-pleural consolidations in one or two regions. As the condition progresses, lung hepatization appears as a tissue-like pattern (consolidation) often with dynamic air bronchograms and unilateral pleural effusion. These signs are highly sensitive and specific for lobar pneumonia [60, 61].

Viral pneumonias and ARDS, by contrast, present bilaterally with B-lines, irregular pleura, sub-pleural consolidations, inhomogeneous distribution, spared areas, and common posterior involvement—distinct from cardiogenic pulmonary edema, which shows a more homogeneous B-line pattern, regular pleura, and frequent bilateral effusions [62, 63]. While the etiologic determination of acute respiratory distress syndrome (ARDS) using conventional ultrasound remains highly challenging, artificial intelligence-based analysis of pleural characteristics has shown highly promising results and may represent a future diagnostic approach.

LUS can track lung aeration daily using an aeration score, helping to assess treatment response by observing score reduction [7, 64].

In ventilator-associated pneumonia (VAP), LUS interpretation is more complex due to factors like atelectasis, previous infections, or ARDS. Posterior consolidations are common in ventilated patients, making them sensitive but non-specific for VAP [65]. A sudden increase in the aeration score may suggest a superinfection [7, 66, 67]. Sub-pleural consolidations in two or more areas can support a VAP diagnosis but are less reliable in interstitial pneumonia or ARDS [65, 67]. An enhanced lobar vascularization indicates the absence of a hypoxemic vasoconstriction, having a high diagnosis sensitivity for VAP [68]. The most specific LUS sign of VAP is a dynamic linear/arborescent air bronchogram within a consolidated area [65, 67]. If the air bronchogram is static or absent, atelectasis cannot be ruled out; bronchoscopy and subsequent modifications of the ultrasound pattern may help clarify the etiology of the tissue-like pattern. [69]. Combining ultrasound with clinical signs in scoring systems improves bedside decision-making [65, 68]. LUS also helps monitor VAP treatment response and guides antibiotic discontinuation [70].

ARDS: diagnosis and assessment of morphological extension of lung involvement Modern LUS relies on three core patterns: A-lines (or absence of B-lines), interstitial pattern (multiple bilateral anterior B-lines), and consolidation pattern. These indicate varying degrees of alveolar loss of aeration but are not disease-specific on their own, as both B-lines and consolidations appear in many lung pathologies [71].

In ARDS, LUS typically shows patchy bilateral findings—a mix of B-lines and consolidations with spared areas—and is often more sensitive than chest X-ray. Recently, LUS has emerged as a valid chest imaging diagnostic criterion for ARDS [72], specifically when bilateral pulmonary abnormalities are identified by an experienced operator. Thus, LUS may eventually be used in place of radiographs for ARDS diagnosis when combined with anamnestic, timing, oxygenation and ventilation criterion.

LUS is also a powerful bedside monitoring tool in ARDS. A 12-zone scan can quantify aeration loss using a score from 0 to 3 per zone by following the progression of A-lines, separated B-lines, coalescent B-lines, consolidation. Recently, a new expert consensus has reached agreement that the aeration score could be further improved by incorporating the percentage of the scan occupied by B-lines [7]. This LUS aeration score is correlated with tissue density, as evaluated by CT scan, and inversely correlated with compliance [73]. Thus, it may help guide PEEP settings, assess response to therapy, and support decisions like prone positioning [74]. Anterior zone scores help distinguish focal vs. non-focal ARDS, influencing management strategies and especially decision on recruitment maneuvers [75]. However, when monitoring a patient using these scores, it is essential to consider the mode of mechanical ventilation—including non-invasive ventilation—as it directly influences lung volumes and, consequently, lung density. In ventilated patients, hypoxic vasoconstriction and high end-expiratory pressure may increase right ventricle afterload and subsequently induce acute right ventricle failure (acute cor pulmonale). Echocardiography helps to early detect acute cor pulmonale, especially in case of high positive end-expiratory pressure (>10 cmH_2O) and/or low PaO_2/FiO_2 ratio (<150).

The LUS aeration score can also be used to predict weaning failure from ventilation [76]. To this aim, LUS is even more effective when combined with cardiac and diaphragmatic ultrasound [76].

How to diagnose and quantify pneumothorax Pneumothorax (PNX) is a potentially life-threatening condition that requires rapid bedside diagnosis and treatment. In the context of traumatology, chest X-rays are performed in the supine anteroposterior position (rather than upright posteroanterior view), which limits diagnosis accuracy. Chest X-rays can, therefore, lead to diagnostic delay and may miss up to 70% of traumatic pneumothorax cases, including 5–7% of large pneumothoraxes requiring chest drainage. Consequently, LUS has emerged as the preferred first-line diagnostic tool in these settings [77].

In PNX, air in the pleural space blocks lung visualization. So, detecting lung sliding, lung pulse, B-lines, or consolidation rules out PNX with high negative predictive value [78]. The lung point is the only pathognomonic sign, but it may not be visible in complete PNX [79]. Studies and meta-analyses confirm that LUS is more sensitive and equally specific as chest X-ray for PNX diagnosis [80].

LUS is effective in post-procedural and perioperative settings (After bronchoscopy or central line insertion) and better detects resolution of PNX after drainage than chest X-ray [81–83]. Although ultrasound guidance is not routinely used for pleural effusion drainage, small studies suggest that it may be useful for locating the drainage site, identifying diaphragmatic excursions and intercostal arteries, and determining the final position of the drain. Lung ultrasound can semi-quantify pneumothorax as large (lung point posterior to the mid axillary line) or small (lung point anterior to the mid axillary line) [84].

Training is limited by the rarity of PNX cases.

How to diagnose and quantify pleural effusion Pleural effusion is a very common condition in ICU patients, affecting up to 83% of those with ARDS [85]. LUS is a key tool for detecting, characterizing, and managing pleural effusions, offering greater diagnostic accuracy than chest X-rays, especially when compared to CT [86]. A 3-h training program with hands-on practice is usually sufficient to gain proficiency in detecting and estimating effusions [87].

In the first diagnostic approach, LUS should be performed using a low-frequency curvilinear or phased array probe, focusing first on the dependent (posterior-lateral) chest areas and directing the probe cephalad [88]. In critically ill patients, it is important differentiating simple and complex effusions. Simple effusions appear as dark (anechoic) fluid and can be transudative or exudative [89] (Video S10). Complex effusions show internal echoes, septations, or loculations, suggesting exudates or malignancy (Video S11).

Many formulas exist to estimate effusion volume, but most show poor correlation with actual drained volume [84]. The Balik's formula (volume = maximal distance between parietal and visceral pleura (mm) × 20) provides easy quantification of pleural effusion [90], and it has been externally validated [91]. While not externally validated, we propose using the formula by Remérand et al. [92]. In ICU patients in the supine position, the effusion volume calculated by CT ($r = 0.903$) and the drained volume ($r = 0.839$) correlated well with the product of the maximal craniocaudal distance on the lateral chest wall (in cm) and the mid-point paravertebral effusion area (in cm^2).

In pleural effusion of critically ill patients, thoracentesis should be considered based on individual risks and benefits [86]. Drainage is associated with an improved PaO_2/FiO_2 ratio and an increase in ventilator-free days. Factors associated with improving outcomes following drainage are: large effusion volume, poor respiratory tolerance, rapid effusion onset, and early drainage. Ultrasound-guided thoracentesis is safe, even in ventilated patients, with a complication rate < 2% [87, 88]. Ultrasound helps find the safest puncture site and reduces risks of pneumothorax or vessel injury. It can also speed up confirmation of lung re-expansion and safe chest tube removal [89, 90].

Future direction: technical innovations, automated measurements, and artificial intelligence

Artificial intelligence (AI) and new technologies are deeply transforming thoracic ultrasound in the ICU. AI currently plays two key roles: automating tasks like guiding image acquisition and classification, and aiding diagnosis by detecting clinically relevant patterns. In cardiac ultrasound, AI supports tasks such as image segmentation and may improve measurements, including left ventricular ejection fraction, IVC collapsibility and sub-aortic VTI [93, 94]. While good image quality is essential and some limitation in tracking sub-aortic VTI, AI can match expert-level accuracy in selected tasks [95].

In LUS, AI-powered models accurately detect various lung conditions [96]. Among the many growing applications, AI demonstrated 92% accuracy in identifying pleural effusion, a high sensitivity for diagnosing pneumothorax through the automated detection of lung sliding and the possibility to standardize the B-line quantification, helping for both aeration and congestion quantification [97–100]. AI also shows a high sensitivity for diagnosing pneumothorax through the automated detection of lung sliding or by quantifying lung sliding with Speckle Tracking technology [101, 102]. Limitations of LUS include reproducibility and comparability as LUS use various scores which are numeric approximation and not quantitative metrics. By reducing inter-operator variability, AI has the potential to make interpretation more reliable and accessible, especially for non-expert users [103–105].

Educationally, AI can be used as a digital tutor, highlighting anatomy and pathology in real-time for novice users, especially when expert mentors are not available [106].

Innovations in the technology of machines and hardware may also support this shift. Handheld devices integrating AI provide rapid bedside assessment, especially useful in resource-limited settings. Robotic ultrasound systems can autonomously scan the chest, reduce staff exposure, which may be particularly useful during pandemics, and may improve consistency and reproducibility

[107]. Wearable ultrasound devices are also emerging, enabling continuous, non-invasive monitoring of heart and lung status [108].

Conclusion

Thoracic ultrasound is now an essential addition to clinical examinations for cases of shock and/or acute respiratory failure. It enables rapid and accurate diagnoses and therapeutic decisions at the bedside. The combination of artificial intelligence with portable and automated new ultrasound technologies promises faster, more objective, and widely accessible thoracic ultrasound in critical care settings. A survey published in 2015 showed that ICU physicians mainly performed echocardiography and few lung ultrasound exams [109]. The current literature now suggests that, in ICU, each echocardiography should be completed by a lung ultrasound and inversely. On an educational point of view, the systematic coupling of both cardiac and lung ultrasound can be summarized by the term "thoracic ultrasound".

Supplementary Information

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Declarations

Conflicts of interest

LM and LZ declare having received fees from General Electric Healthcare for ultrasound lectures. MG is a consultant for General Electric Healthcare's point of care ultrasound group. RA is founder and share holder in Deep Breathe Inc, an AI ultrasound company. Other authors did not declare any conflict of interest.

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